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CLINICAL TREATISES

on the

SYMPTOMATOLOGY *and* DIAGNOSIS

of DISORDERS *of* RESPIRATION *and* CIRCULATION

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Professor of the Second Medical Clinic, Vienna; Associate Editor
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PART III

ANGINA PECTORIS

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PREFACE TO AMERICAN EDITION

Angina Pectoris, on account of its high mortality and sudden onset, and because, too, it has afflicted so many distinguished men, among whom may be mentioned Seneca, John Hunter, Arnold of Rugby, has always aroused the intense interest of clinicians.

Professor V. Neusser in the present volume has presented very lucidly and succintly his view-point, supported by a great personal experience and vast clinical material.

His conclusions should receive the careful consideration of thoughtful practitioners, and are of great value as throwing some light on a disease still the subject of speculation.

No attempt has been made to add to the original as the subject matter is so personal that additions would have marred the harmony of this medical classic.

Andrew MacFarlane.

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Angina Pectoris

I

DIAGNOSIS OF ANGINA PECTORIS

The diagnosis of "angina pectoris, caused probably by an affection of the coronary arteries," is never in doubt, when an elderly person suddenly experiences a more or less marked pain behind the sternum and in the precordial region with intense anguish. The pain radiates in different directions, especially toward the left side of the neck and left arm, and sometimes toward both extremities and the shoulders. The patient is perfectly conscious, experiences no difficulty in breathing, becomes pale, his forehead covered with cold perspiration, and his face Hippocratic. The paroxysm either passes over after a short time with a retrogression of the above symptoms, or the patient dies during the attack. Although the diagnosis of pronounced stenocardiac attacks is easy, it presents considerable difficulty in rudimentary seizures or if the pathological picture becomes obscure through accessory symptoms which, as a rule, are absent in typical cases. The difficulty of forming a correct diagnosis in these rather frequent cases is due to the fact that angina pectoris is not a disease in itself, but only a symptom, or rather, a symptom-complex. This difficulty is similar to that in an epileptiform attack. It is impossible to tell at first glance whether it is caused by a brain tumor, syphilis, cysticerci, intoxications, sclerosis of the cerebral vessels, etc., or whether it is genuine epilepsy. So it is equally difficult to establish a precise causal diagnosis from the symptoms present in complicated cases of angina pectoris.

In these obscure cases it is not only necessary to analyze the various components of the stenocardiac attack, but also to make a careful examination of the patient. The history in regard to both the attack itself and the previous diseases of the patient is of particular importance.

Cardinal Symptoms.—The cardinal symptoms of angina pectoris include retro-sternal pain and the fear of impending death. The intensity of the pain may vary; in some cases it is violent, constricting, piercing, stabbing; in others it is hardly perceptible. The patients have the sensation of death supervening at any moment. This dread of impending annihilation, and the pain, may be so intense in the beginning of an attack that the sufferer utters a loud, piercing shriek.

Retro-sternal Pain.—The location of the pain varies; in most cases it is behind the sternum, to the right or to the left; sometimes higher, sometimes lower, and sometimes in the middle of the sternum or in the region of the xyphoid process. These cardinal symptoms, however, show many variations. There are patients in whom the feeling of oppression and anxiety, with pallor of the face, occupy the foreground of the picture, and they die at the beginning of this feeling of oppression without complaining of any particular pain at all. In other cases the pain is the predominant symptom, while the collapse is only indicated by a faint pallor of the face. The intensity of the pain is not always proportionate to the gravity of the attack; in fact, in the very gravest forms of coronary angina there may be almost total absence of pain. A patient once came to Potain complaining of slight thoracic distress, to which however, he did not attach any particular attention: during the examination he fell down dead.

Radiation of Pain.—Another peculiarity of stenocardiac pains is their radiation in definite directions, especially toward the left side of the neck and the left arm, sometimes down to the third and fourth fingers, along the course of the internal cutaneous and the ulnar nerves. After the attack a sensation of numbness and tingling remains in the fingers for a time. Frequently the radiations occur in both brachial plexuses, sometimes only to the right arm, quite irrespective of whether the retrosternal pain is on the right or left side and whether there is a constriction of the right coronary artery or whether the left one is affected. It has also been observed that the pain may radiate to quite remote nerve areas. Thus, for instance, the dorsal pain may commence in the region of the first dorsal vertebra and be diffused into the precordial region; it may originate in the lower extremities, the lumbar nerves and even the hypo-gastric region, the testicles or anus, and radiate upward to the region of the trigeminus, so that during the paroxysm, the pain is regarded as dental or maxillary.

Unusual Radiations of Pain.—In other cases the radiation to the shoulders and arms is absent, instead of which intense pain is felt in the abdominal organs, manifesting itself as severe gastralgia or intestinal colic. Violent gastralgia with collapse may be the only and terminal manifestation of an attack of angina pectoris.

All these abdominal forms of stenocardia must be looked upon as radiations along the course of the pneumogastric nerve and the sympathicus abdominalis. They are accompanied by vagus manifestations on the part of the esophagus (dysphagia, nausea) and disturbances of the larynx (aphony, hoarseness), while the vasomotor symptoms, perspiration, coldness of the extremities, dilatation of the pupils, should be looked upon as manifestations connected with the sympathicus.

The polymorphic character of these radiations, which may in many cases occupy the foreground of the clinical picture, may conceal the real affection beyond recognition. The gastralgic forms may be disguised as gastric ulcers, dilatation or catarrh of the stomach, enteroptosis or aneurysm of the abdominal aorta, until sudden syncopal death supervenes and the abdominal form of angina discloses itself as a true coronary angina. Radiation to the hepatic region may be mistaken for cholelithiasis. I remember the case of a young married woman, suffering from aortic insufficiency with stenocardiac paroxysms, who had a subicteric color following such an attack, accompanied by vomiting, with an enlarged liver which was very sensitive to pressure. A diagnosis was made of cardiac insufficiency complicated by cholelithiasis. At autopsy no gallstones were found, but narrowing of the coronary arteries. Cases have also been reported where nephrolithiasis has been mistaken for angina pectoris on account of the pains radiating into the lumbar region and the testicles. In patients with cardiac insufficiency these lumbo-abdominal neuralgias may, however, have a different cause and even be the sequel to a renal infarction. In its further course paranephritis may develop around the infarction, culminating in connective tissue proliferation. In these cases the ileo-hypogastric nerve may become compressed or even embedded in the connective tissue proliferation with a resulting severe neuralgia. This was observed by Curschmann, especially in patients suffering from aortic insufficiency. Thus there is a veritable labyrinth awaiting the diagnostician, who is confronted with the question whether he has to deal with angina pectoris with radiations to the lumbar plexus or with angina pectoris complicated by previous adhesive paranephritis, or, finally, with a complication of nephrolithiasis.

Heart Action and Pulse.—The cardiac action and pulse may show different characteristics during the attacks. In many cases the action is extremely stormy and rapid, from 150 to 200 beats; in others the contrary is present, the cardiac action being reduced to 28. In the latter case the patient has the sensation as if his heart stood still. The condition of the pulse is also variable; sometimes it is small, soft and easily compressible, or small and tense; at times it is regular or irregular. It is an important fact that even in grave attacks of angina pectoris there may be perfectly normal cardiac action and pulse; in these cases the behavior of the pulse and heart is precisely the same before and during the attacks. It is important to note that in patients with cardiac insufficiency, the arrhythmia which exists during the intervals, generally disappears in the attacks, and that heart murmurs which were formerly present cannot be observed during the paroxysm. A surprising smallness of the left radial pulse as compared with the right has been repeatedly observed during stenocardiac attacks. This behavior of the left radial pulse does not justify the diagnosis of aneurysm or atheromatous stenosis of the left subclavian, not even in case this symptom persists in the intervals between the paroxysms. There may be a congenital anomaly of the vessels.

Respiration Unaffected.—A further important sign of the stenocardic paroxysms is the integrity of respiration. In pure, uncomplicated cases of angina pectoris a characteristic sign of the attack is not dyspnœa, but, on the contrary, apnœa, a fact specially dwelt upon by Huchard. In consequence of the violent pain, patients suppress every muscular movement and frequently inhibit their breathing after a deep inspiration in order to immobilize the thorax. It is through the absence of difficulty in breathing that a stenocardiac paroxysm is distinguished from both bronchial asthma and the asthmatic attack of patients with cardiac disease.

Respiration Affected.—Cases, however, are not infrequent, especially after prolonged existence of the stenocardia, where there is a complication of angina pectoris with cardiac asthma. In such cases the pulmonary manifestations are occasioned by the cardiac insufficiency, while the angina pectoris, like the asthma, is a secondary condition to the original affection, namely, arterio-sclerosis. Cyanosis and pulmonary edema also occur with their characteristic bloody expectoration, especially in protracted attacks of angina pectoris. That the latter affection and asthma are independent of each other, is proved by those cases in which the stenocardiac attacks alternate with the asthmatic.

Complicating Myocarditis.—Of special interest are those cases of stenocardia in which, with the onset of broken compensation, angina pectoris disappears to make room for chronic dyspnœa with its well-known signs of broken compensation. The subsidence of the angina may be due to the development of a myocarditis or an insufficiency of the mitral or even of the aortic valves, following a previously existing arteriosclerosis of the aorta. Some authors look upon this as "a self-cure" of the organism. Musser has seen the symptoms in several cases of angina pectoris disappear after the signs of cardiac dilatation had developed, the absence of stenocardiac attacks persisting for years, as long as there was dilatation of the heart. With diminished dilatation the crises of angina pectoris may reappear. Musser goes so far as to pronounce a more favorable prognosis in angina pectoris if there is simultaneous dilatation of the heart; the latter, on the other hand, is prognostically less favorable if there is a simultaneous angina pectoris. In this respect, and from a theoretical point of view, cardiac dilatation would indicate a "noli me tangere," especially in regard to the administration of digitalis. It is certainly true that cases occur, where, with an improvement of cardiac function after the administration of digitalis, the stenocardiac attacks become more frequent; but on the other hand cases are not wanting which prove the opposite.

High Blood Pressure.—These facts impel us to discuss more in detail the relation of angina pectoris to blood pressure. The favorable effect of the nitrites, especially of amylnitrit and nitroglycerin, which are known to have a vaso-dilating effect, readily suggested the assumption that angina pectoris was dependent upon vascular spasm. This is especially true of the nervous forms of angina pectoris, of which Nothnagel's angina vasomotoria must be regarded as the prototype. Huchard speaks of an arterial hypertension as an important symptom which precedes or accompanies most stenocardiac attacks. According to Mackenzie the preponderating majority of paroxysms of angina pectoris are caused by cyclic increase of the blood pressure and periodical overstraining of a weakened heart; "the paroxysm is caused in a predisposed heart by the fact that there is increased resistance to the cardiac function." Pal also looks upon high blood pressure as a factor favorable to the occurrence of stenocardiac paroxysms. He observed cases where the patients suffering from stenocardiac attacks did not show any increased pulse tension during the free intervals, but a considerable increase of the blood pressure during the paroxysms. On the other hand numerous cases have been reported where the blood pressure decreased during the stenocardiac paroxysms. We are able to report a few interesting observations of this kind:

1.—Hermina B., 36 years of age, no occupation. Married 12 years, no children. Husband healthy. Has had cardiac distress for three years, palpitation after fast walking. During the last six months there have been stenocardiac paroxysms with pain radiating into the left arm. In October, 1903, there were numerous attacks, as many as twenty a day, and there were also attacks during the night when turning over in bed. The heart is enlarged toward the left, there are systolic and diastolic murmurs over the aorta and a systolic thrill is palpable. There are no murmurs at the cardiac apex. Slight dullness over the aorta. Tonometer 130.

Diagnosis:—Insufficiencia et stenosis aortæ, dilatatio aortæ levioris gradus.

In November, 1903, after the administration of 3.0 grams of diuretin daily, the frequency of the paroxysms was reduced to two or three a day. In December, 1903, 2.0 to 3.0 grams natrium iodatum per day were administered, without any effect upon the stenocardiac attacks or on the blood pressure. Blood pressure 165. About four stenocardiac paroxysms occurred in a day. Rhodannatrium and Marienbad water were given without effect. In January, 1904, the blood pressure was 150. Laxative treatment, senna with manna and Jalap, also rest in bed, reduced the blood pressure to 135. The stenocardiac paroxysms, however, continued unchanged both in frequency and intensity. In March, 1904, the blood pressure again registered 150. The attacks were reduced to two a day by the administration of diuretin.

2.—Karl P., 33, barber. Father died of pulmonary tuberculosis. Mother and three other children healthy. In January, 1898, contracted a soft chancre with suppuration of the inguinal glands. In November, 1902, patient, after having partaken of three glasses of beer in the evening, experienced on his way home for the first time violent pains on the left side of the chest, accompanied by trembling of the entire body. Later on the pains recurred at irregular intervals, especially when walking, and they radiated toward the left axilla and afterwards also to the left shoulder and arm down to the tips of the fingers, while the patient had the sensation of considerable blood pressure toward the head. These attacks were frequently accompanied by violent eructations, passage of flatus and rumbling in the abdomen. The paroxysms lasted some minutes, sometimes half an hour, during which there was anguish and agony as if the heart were being compressed. The face during these attacks was greatly congested. The attacks occurred principally when bending forward, walking rapidly and after prolonged work. The patient is a beer drinker and a moderate smoker.

Physical condition:—Patient is well nourished, has some facial congestion, pulse of the radial and carotid arteries remarkably tense, although there is no pulsation in the dorsal arteries of the feet and in the crural arteries. There is a slight dullness of the manubrium sterni and the cardiac dullness extends to the right sternal border. The cardiac impulse is palpable within the mammillary line. A systolic murmur is heard at the apex and becomes progressively louder toward the base; the second aortic sound is accentuated.

Findings at the x-ray examination: Dilatation of the aorta with distinct pulsation at the level of the third intercostal space. Ventrodorsal translumination shows a diffuse shadow running obliquely downward from the center to the left side, which also shows pulsation. It is impossible to follow the aorta lower down beyond the dilatation.

Diagnosis:—Stenosis of isthmus with dilatation of the ascending aorta. Angina pectoris.

Treatment:—Natrium iodatum and Rhodannatrium, 2.0 grams per day, without success. After the administration of diuretin, 3.0 grams per day, the paroxysms become less frequent. The blood pressure on both sides, in January, was constant at 190 to 195. In spite of the administration of 600 grams of natrium iodatum in four weeks the blood pressure remains unaltered, also the stenocardiac attacks. In June, 1903, the blood pressure was 195. After the administration of 3.0 grams of diuretin per day the attacks decreased in frequency, but the blood pressure remained unchanged. In November, 1903, treatment with Marienbad water was commenced and continued for six weeks, after which the blood pressure sank to 190. In February, 1904, venesection was performed (removal of 150 c.c. of blood), upon which the blood pressure sank to 160, but rose again after the lapse of a week. In spite of the diminished blood pressure the painful paroxysms are not affected. Diuretin and nitroglycerin alone are able to relieve them, but without influencing the blood pressure.

A third case of angina pectoris was observed in a patient with atheroma and dilatation of the ascending aorta. Ten attacks occurred in twenty-four hours, even after slight exertion, and ceased in a few minutes as soon as the patient rested. The tonometer registered regularly 170 to 175. When diuretin was administered, 3.0 grams daily, the paroxysms diminished in both frequency and intensity.

These three observations, therefore, show that there is not necessarily a parallelism between frequency and intensity of the stenocardiac paroxysms and the blood pressure; that a reduction of the blood pressure did not affect the angina pectoris; and that diuretin did frequently reduce the number of attacks without changing the blood pressure.

Low Blood Pressure.—More instructive still are those cases of angina pectoris which occur with low blood pressure and are improved by the administration of digitalis, which increases the blood pressure. This is illustrated by the following case:

3.—Francis P., 40 years of age, contracted in 1880-81 a severe acute articular rheumatism. In 1891 he contracted a venereal affection with considerable swelling of the prepuce, ulcers about the glans penis and suppuration of the left inguinal gland. Since 1895 he has suffered from paroxysmal pains in the pit of the stomach which, on rapid walking or climbing, increased to such an extent that he had often to stand still and wait until the attack subsided. The pains frequently occurred after the ingestion of food, the quality of which did not materially influence the severity of the attacks, nor did change of position. Appetite was good, but during the paroxysms there were eructations and sometimes vomiting. In the further course, the painful attacks which were originally confined to the epigastrium, underwent a change. A burning sensation appeared in the cardiac region which often, on occasions of unusual severity, radiated to both shoulders, to both upper extremities and down to the tips of the fingers. This radiation was generally unilateral, either into the left or the right arm, very rarely into both simultaneously. The attacks lasted ten to fifteen minutes and were not associated with either anxiety or any particular pallor of the face. There was no dyspnœa worth mentioning. These attacks had persisted for almost nine years with more or less improvement. The patient, an inspector in the military finance service, had to apply for an easier post, as the exertion of walking and climbing increased the attacks. Since October, 1903, the painful attacks increased both in frequency and intensity, considerable dyspnœa manifested itself, and latterly, also œdema of the lower extremities. The examination disclosed greatly intensified cardiac impulse in the seventh intercostal space, distinct pulsation in infrasternal depression and the suprasternal notch, considerable increase of the cardiac dullness and a loud diastolic murmur at the aorta. The gastric region was somewhat distended and sensitive to pressure; liver and spleen not enlarged.

Diagnosis.—Insufficiency of the aortic valves with considerable hypertrophy of the left ventricle.

After entering the clinic, the patient had, on March 11, three paroxysms of retrosternal pains of fifteen minutes' duration, each without experiencing any special feeling of anguish. Pulse 96, and regular, respirations 25. On March 14 the blood pressure was 90. On March 15 a severe stenocardiac attack of short duration, with vomiting. Blood pressure equally low during the attack. After the administration of digitalin with Theocin for several days the blood pressure rose to 110 and the stenocardiac attacks ceased, but the dyspnœa and cephalalgia persisted. With increased weakness, cyanosis, bloody sputum and manifestations of a pulmonary infarct, the patient expired.

Autopsy.—Long standing destruction of the right aortic segment from ulcerative endocarditis with consequent marked atheroma of the thoracic aorta; considerable excentric hypertrophy of both sides of the heart; atheromatous stenosis at the beginning of the coronary artery; thinning of the cardiac wall corresponding to the right apex, thrombosis of the right auricle; multiple pulmonary infarction; considerable congestive induration of the organs.

This case illustrates that stenocardiac attacks may occur even with low blood pressure, and that increased blood pressure under the influence of digitalis did not increase, but rather diminished, the frequency of the painful attacks. From these observations it follows that increase in blood pressure and angina pectoris do not stand in the relation of cause and effect, and that the increase in blood pressure preceding the attacks, which has been observed during the paroxysms, is connected with the original pathologic cause of the angina pectoris, while the increased blood pressure which has been observed during the paroxysms is possibly owing to the attacks of pain and the consequent reflex irritation of the vasomotor center. The increase in the blood pressure in these cases would, therefore, not be the cause, but the consequence of the stenocardiac paroxysms. In cases associated with cardiac weakness from diseased coronary arteries or preexistent degeneration of the cardiac muscle the pulse at the beginning of a stenocardiac attack will be soft, easily compressible and of low tension. The forms, in which the cardiac insufficiency occupies the foreground, are frequently complicated by cardiac asthma and may go on to a fatal termination with manifestations of cyanosis and pulmonary œdema. In this asphyctic form of angina pectoris Huchard does not consider the pain, the object of treatment, but the cardiac weakness. Morphin is not indicated, but rather excitants such as camphor, ether, spirits of nitrous ether, etc.

Since there are cases of angina pectoris which are independent of the blood pressure and since, on the other hand, stenocardiac attacks in aortic patients will persist even if cardiac insufficiency and with it decreased blood pressure develops, it is clear that in the treatment of these conditions digitalis will retain its place and that an arbitrary exclusion of digitalis from the therapy of the treatment of angina pectoris is not permissible.

Variations in Blood Pressure.—The condition of the blood pressure in angina pectoris, therefore, depends chiefly on the localization of the vascular spasm. If the latter involves the region of the splanchnics, it can easily be understood that with unchanged cardiac function a marked increase of the blood pressure may take place. If the spasm is localized in the thoracic region, the paroxysms may be accompanied by a vasoconstriction of the vessels of the upper extremities and of the region of the coronary arteries. The intensity of the manifestations will depend upon which of these factors prevails. The limitation of the spasm to the region of the coronary arteries reduces the nutrition of the heart, and the consequent reduced action of the heart effects a decrease in the blood pressure.

II

ETIOLOGY OF ANGINA PECTORIS

Arterio-sclerosis.—The etiology of angina pectoris is just as polymorphic as its symptomatology. The most frequent and best known causes of angina pectoris are organic affections of the aortic valves and of the aorta itself, which are accompanied by stenosis of the coronary arteries. This form of angina pectoris is found in arterio-sclerosis, affecting either the point of origin of the coronary arteries or their course: also in fatty heart, gout, diabetes and tabes, in many cases of chronic lead and tobacco poisoning, probably also in syphilis where the involvement of the vessels forms the connecting link between angina pectoris and the original affection. Angina pectoris in aneurysms of the aorta also belongs to this group, inasmuch as the latter are developed on an arterio-sclerotic base. Here also belong those cases of angina pectoris in which extensive adhesions of the heart to the pericardium constrict the lumen of the coronary vessels. These anatomical findings which are recorded in the preponderating majority of grave cases of angina pectoris, form the basis for the arterial theories, of which the French clinicians, Potain and Huchard, are regarded as the chief representatives.

Of particular interest are those cases where relatively young patients died suddenly after stenocardiac paroxysms and where the autopsy proved the coronary vessels as well as the aorta in their course to be almost intact, but revealed at the beginning of the right or left coronary artery very small atheromatous plaques which had occasioned the occlusion of the vessels.

Ischæmia of the Myocardium.—As a result of this arterial theory, every case of angina pectoris was considered to have been caused by ischæmia of the myocardium. The quantity of blood coursing through the constricted vessels may be sufficient for the normal requirements of the myocardium; if, however, the heart is called upon to perform more work in order to overcome the increased blood pressure, the quantity of blood contained in the vessels will not suffice to nourish the heart, and there will be developed the symptom-complex of angina pectoris in consequence of deficient nutrition of the heart.

Clinical observation, especially the occurrence of paroxysms after exertions, notably after eating, walking against the wind, exposure to cold weather, climbing stairs, coitus, defecation, great excitement, all of which are factors which raise the blood pressure, favor the assumption that angina pectoris is caused by anischæmia of the myocardium. The fact that in autopsies considerable stenosis of the vessels was found in elderly individuals who during life never displayed any symptom of angina pectoris, and again the experience that in many cases the pulse was found to be absolutely normal, compels us to assume that the mechanical constriction of the vessels cannot be the sole cause of the stenocardiac attacks.

Active Vasoconstriction.—We have rather to assume, as did Erb in regard to dysbasia intermittens arterio-sclerotica, that in addition to the constant factor of anatomical constriction of the lumen there is another inconstant factor, namely, active vasoconstriction due to hypertrophy of the muscularis or to exaggerated excitation of the nerves and ganglia in the vascular wall, which favors the occurrence of the functional disturbance. Arterio-sclerosis of the lower extremities causes no pain in spite of the constriction of the lumen, and pain only occurs as the ischæmia increases from vasoconstriction. In the same way, we must assume that an abnormal vascular spasm, added to the constriction, is responsible for the pains in coronary angina. It is, of course, presupposed that the vascular wall still possesses a sufficient degree of elasticity for vasoconstriction to have an ischæmic effect. If, however, the vessel be changed into a rigid tube, the former stenocardiac attacks may cease and the unchanged vascular stenosis may manifest itself clinically as insufficiency of the myocardium. This is observed in senile hearts and in many cases of angina pectoris as the atheromatous process progresses into the coronary arteries. If, as sometimes happens, the paroxysms are relatively rare in angina pectoris, an increased frequency of the attacks may render the vasomotors hyperesthetic and in the further course of the disease may cause spasms when there is but slight excitation. In cases, however, where the functional factor is of less importance, a relatively strong irritation may be necessary to induce pain when the anatomical lesion progresses. These cases are gravest and usually end fatally. Generally speaking, it may be stated that the less frequent, the graver are the attacks. If grave attacks follow in rapid succession, the worst prognosis would be justified.

The assumption that angina pectoris with coronary changes occurs in conjunction with arterio-sclerosis of the aorta is supported by the experience that in other affections of the heart and cardiac valves the attacks occur only very rarely.

Endocarditis.—In Endocarditis of the mitral or even aortic valves paroxysms resembling stenocardia are almost never observed except when the valves are torn. On the other hand, the occurrence of true paroxysms of angina pectoris in endocarditis vegetans is easily understood from the occlusion of the coronary opening, and their mechanism is the same as that of atheromatous stenosis of the opening. In aortic insufficiency of rheumatic origin, in which the aorta itself remains intact, angina pectoris is an occurrence of the greatest rarity: a fact which should not be underestimated for purposes of differential diagnosis. In mitral stenosis and mitral insufficiency, angina pectoris is almost unknown, the painful sensations in the cardiac region in severe mitral stenosis occurring chiefly in nervous and hysterical individuals. The thrombi, which in the severest forms of mitral stenosis occur in the extremely dilated left auricle, present, in spite of simultaneous ischæmia of the aorta and the coronary arteries, a picture of acute cardiac weakness with cyanosis and dyspnœa, rather than that of angina pectoris. The same holds good for the affections of the tricuspid valve; even in stenosis of all the three ostia (mitral, tricuspid and aortic) of which we observed several examples in our clinic, nothing in the nature of stenocardiac attacks has been noted. The precordial pains occuring at the moment of fatal syncope in the infectious forms of myocarditis (diphtheria, variola and typhoid), materially differ from typical angina pectoris as do the cardiac symptoms which occur in rupture of aneurysms or of the heart. The accompanying pain and dyspnœa are much more readily explained by the compression of the heart and phrenic nerve as well as the adjacent pleura by the hematoma. Since aneurysms of the heart and of the aorta can be traced back to arterio-sclerosis, a complication of these conditions with coronary angina is easily understood, and an individual who has suffered from stenocardia may suddenly die of heart rupture.

Pericarditis.—Pericarditis stands in quite a peculiar relation to angina pectoris. Acute effusions into the pericardial sack may, similarly to hematoma, be accompanied by a dull precordial pain, together with dyspnœa and cyanosis. In many cases the pains are occasioned by neuritis of the phrenic nerve, a fact which French clinicians especially have emphasized and which we ourselves have also observed. We have previously called attention to the relation of angina pectoris to adhesions of the pericardial layers and to the possibility of stenocardiac attacks in conjunction therewith, owing to the constriction of the branches of the coronary arteries. It is nevertheless surprising that in extensive adhesions in tuberculous pericarditis stenocardiac attacks are seldom observed, whereas cases of postrheumatic pericardial adhesions show relatively often the symptom complex of angina pectoris. It is possible that in some cases the localization of the anatomical lesion at the point of origin of the large vessels, involving the periaortic and coronary plexus, exerts influence on the severity of the stenocardiac attacks. There are, however, rare cases of angina pectoris leading to sudden death, in which an autopsy reveals only an adhesion between the heart and pericardium without stenosis of the lumen of the coronary vessels.

Auscher describes the case of a man 50 years of age who, two years after rheumatism, fell ill, showing the typical manifestations of angina pectoris, suffered for several years from this affection and died during one of the attacks. At autopsy adhesions of the heart to the pericardium were found, while the aorta and coronary arteries were absolutely normal at their origins. A patient of Fraenzel, 48 years of age, suffered, without known cause, an exquisite attack of angina pectoris one morning when preparing for his usual walk to the office. This was repeated each time he ventured into the street so that he grew afraid to go out unaccompanied. One year after the first attack, at the age of 49, he died from erysipelas of the head after a three days' illness. His heart was found intact, only the musculature was somewhat soft and friable and the coronary arteries were absolutely unchanged.

Dilatation and Fatty Degeneration of the Heart.—Cases of angina pectoris from over-exertion of the heart, dilatation and fatty degeneration, where mere movement gave rise not only to stenocardia, but also to dyspnœa, cough and sometimes hemoptysis, are rare in medical literature, but owing to the fact that autopsy in these cases disclosed the coronary arteries to be intact, they possess a certain significance for the pathogenesis of angina pectoris. It will be observed that not only these cases, but also those of pericarditis with anatomically intact coronary arteries, may present during life the symptomatic picture of angina pectoris. It is just these and similar cases which formed the foundation for the neuritic theory of angina pectoris. It was assumed that an inflammatory affection of the aortic and coronary plexus may follow in the wake of infectious and other diseases, and Lancereaux has histologically proved such a case.

Plexus Anginas.—In the group of plexus anginas such forms have also been incorporated as were observed in dilatation and aneurysms of the aorta without lesions of the coronary arteries. They involve the plexus by extension of the inflammatory process which had originated in the neighborhood of the aorta. More instructive still are some cases, reported in the older literature, which cannot be easily made to agree with the cardiac theory of angina pectoris. At autopsy Blondin found in the vagus of a person who had suffered from angina pectoris, a tumor the size of a large pea, which consisted of firm, gray tissue and had probably been formed by internal proliferation of the neurilemma. Heine* describes the case of a mason 36 years of age, who had periodical paroxysms of unspeakable anguish precisely as in angina pectoris, during which the cardiac function was completely suspended for four to six beats. The history of the patient was as follows:

Appearance of patient somber and aged. He complained that his heart frequently stood still and that the heart-beat could not be felt for four to six beats at a time. The appearance of the patient during the cessation of the cardiac function confirmed the fact that he experienced terrible anguish. He looked as if thunder-struck, became speechless and motionless. Although fully conscious and sitting upright and unsupported, his wide-open eyes apparently seemed to take in his own terror-stricken picture. The patient declared that he had a presentiment of the imminent cessation of the heart-beat for a second or more before its onset by experiencing a sort of unrest and oppression of the chest; that, as the heart stopped, there was a violent pain, starting from both sides of the thorax and spreading upward toward the nucha and the head, where it remained stationary for some time after the attack passed over. These attacks recurred at irregular intervals, sometimes as often as ten or twelve times a day, then not to return, perhaps, for several weeks. During the last eighteen months they became much more frequent. When the heart-beat returned accompanied by a sigh, no change was noticeable in the patient's condition except that the first few beats followed in more rapid succession. In the interval between the attacks rhythm, tone and size of the heart were normal. The patient coughed, frequently complained of vertigo and there was slight fever toward evening, but never dyspnœa. He was able to lie on both sides, to climb stairs without difficulty, and during the attacks his face never displayed any congestive hyperæmia or dyspnœic blueness, but became pale. Gradually the attacks became more and more frequent and lasted longer; the pain in the neck and head never left him. He grew weaker from day to day, was unable to leave the bed on account of vertigo, and died in a comatose condition which had lasted for several days.

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* Mueller's Archiv f. Physiologie, 1841, p, 236.

Heine, together with Professor Rokitansky, Skoda, Kolletschka and Gutbrod, had repeatedly examined the patient and diagnosed the trouble, in view of the unimpaired respiration and heart action, the unaffected spinal cord and the other cervical nerves, and the absence of any pericarditic manifestations, as cardiac epilepsy consequent upon a pseudoplastic tumor situated either at the upper cervical plexus or at the cardiac nerves, with hypertrophy of the cervical spinal cord.

The autopsy, made by Rokitansky, resulted in the following findings:

"Autopsy Report on the body of Sebastian Wuerschel, mason, 36 years of age, who died in room 52; autopsy made December 18, 1833, Protocol No. 209, sec. 8739.153.

"Body of medium size, emaciated; skin and musculature—especially the latter—atrophied, hair brown, eyes blue, neck thin, thorax flat, abdomen somewhat retracted.

"Cranial Cavity.—The cranial vault is rounded, large, of ordinary thickness, compact, with sutures closed. The inner aspect of the frontal bone is marked on both sides with cribriform pits; the posterior third of the falciform fossa is surrounded by ridgelike borders up to the lambdoid angle. The falciform body contained some fluid blood. The dura mater is very thin, stretched tensely over the bone and its vessels are compressed. Convolutions flattened. The arachnoid in the middle of the lateral edge of both hemispheres marked with a group of granulations the size of a cent, and at the anterior cerebral lobe with numerous Pacchionian bodies. Cervical cortex thin, medulla doughy, lateral cerebral cavities very much distended and contain about four ounces of clear serum; venous plexus covered with vesicles, the size of a millet or pea. The pineal gland is finely granular at its base, left cerebral hemisphere large, at its posterior border along the horizontal part of the transverse sinus sprinkled with numerous yellowish tubercles from a hempseed to a pea in size. The medulla, with the exception of the crus cerebri and corpus quadrigeminum, is softened and of a pale yellowish tint. Both, the vermis cerebri inferior and the medulla oblongata, are crowded to the left, the concavities at the cranial base markedly pronounced and the left posterior fossa somewhat distended. The mucous glands in the anterior lobe are flattened; the posterior lobe is more roomy, round, the color of dark brown, and of pulpy consistency; the cellular stratum connecting the same with the anterior lobe is jelly-like. The anterior aspect of the petrosal bones below the exterior portion of the Gasserian ganglion is unevenly and cribriformly absorbed. Similar areas are on the large wings of the sphenoid, resembling deposits of Pacchionian bodies.

"Thoracic Cavity.—Whitish froth is collected in the trachea. Both lungs, especially the left, are adherent at their apices and the right lung at one place for its circumference. Both are distended, large-celled, hyperæmic, œdematous in places, contracted to a considerable extent at the apex, especially on the left side, into a tough, knotty blackish-blue mass containing cheesy, chalky material. There is a similar place the size of a hen's egg toward the anterior lower end of the upper lobe. The bronchial and lymphatic glands along the internal mammary and behind the larger vascular trunks are changed into bluish-black, tough nodes, interspersed with concrements of lime. Such a node, the size of a bean, interrupts the phrenic nerve (right) in the middle of its course at the base of the lung, and completely interweaves it with the exception of a small remnant of the neurilemma running over its surface, which is thick at the top, but remarkably thin at the bottom.

"Heart.—Flabby. The large cardiac nerve arising from the plexus between the descending aorta and the pulmonic artery is interwoven below its arch with a black body, the size of a hazelnut, and thickened before entering it. The branches of the left vagus, descending along the anterior border of the left bronchus to the pulmonary plexus, are similarly affected by a knotty, bluish-black lymph gland underneath. The pericardium contains two drams of serum, the heart is of normal size, tough and contains fluid blood in the ventricles and large vessels.

"Abdominal Cavity.—The liver is large, coarsely granular, tough. The gall-bladder contains brownish-yellow bile. The spleen is brownish-red and friable. Pancreas soft. The ganglion of the solar plexus is very large, and enveloped in a pale red, vascular, cellular tissue. The stomach is filled with air and a gray, muco-serous fluid; the intestines are in part moderately distended, partly stenosed, and their inner walls covered with a bile-stained mucus, while the mucous membrane below is partly undermined. The large intestine contains hardened fecal matter. The kidneys are hyperæmic and the bladder contains light-colored urine.

"Spinal Cord.—Between the membranes of the spinal cord there is a collection of a grayish serum. The spinal arachnoid is covered with millet to lentil size bony plates, especially in the upper lumbar and dorsal sections. Pia mater hyperæmic, its vessels distended, medulla large and thick, its cervical portion strikingly enlarged, its substance soft, the cortical (gray) mass very pale and the medullary portion dazzlingly white."

Even though this pathological picture, as described by Heine, deviates in many respects from the classic type of angina pectoris, inasmuch as the patient was able to climb stairs without distress, yet the principal symptoms of the attacks, notably the severe thoracic pains radiating toward the neck and nucha, the feeling of oppression, the immobility, the agony, the cessation of cardiac action, the integrity of respiration during the attacks and their short duration, also the pallor of the face, are so pronounced that, on seeing such a patient, we should hardly make any other diagnosis than angina pectoris; and yet the autopsy revealed complete integrity of the heart, so that it can only be assumed that the cause of the attacks observed during life was the anatomical lesion in the area of the nervus phrenicus dexter, of the nervus cardiacus magnus and of the branches of the left vagus descending to the pulmonary plexus at the anterior side of the left bronchus. We have, therefore, to deal with a form of stenocardia, the anatomical substratum of which is to be looked for above the coronary and aortic plexus.

Still more interesting is a case which was reported by Wallenberg in Danzig:

A policeman, 31 years of age, while arresting a disorderly person, was stabbed in the back three times by the companions of the prisoner. The location of one of the wounds was near the process of the third dorsal vertebra and justified the assumption that the third left dorsal nerve had been injured at the spinal ganglion. Soon after there was an intense feeling of anguish in the cardiac region, together with a sensation as if the heart stood still. There was moderate anæmia and bradycardia. The pulse, which was regular, vigorous and full, sank, when patient assumed the recumbent position, on various occasions to 42 beats in a minute, even at a temperature of 38.5 C. Wallenberg, when first examining the patient, thought of the possible existence of a coronary sclerosis before the injury. Upon more detailed examination he found that there was decreased sensibility of the skin between the sternum and the left mammilla and especially along the inner aspect of the left upper arm from the axilla to 9 cm. above the condylus internus humeri. He concluded that both the impaired sensitiveness and the distress owed their origin to the injury of the left dorsal nerve at the point where it leaves the vertebral canal. As the dorsal nerve also contains branches of the sympathetic which go to the aortic and pulmonic plexus and to the thoracic ganglia, it readily suggested itself that there was a causative connection between the injury and the paroxysms as above described, especially as the heart action became normal after the healing of the wound, and, as may be inferred from the history, the patient completely recovered.

Tabes Dorsalis.—Closely allied are the tabetic forms of stenocardia, the etiology of which has not yet been sufficiently investigated. Leyden thought that in the rare cases of angina pectoris in tabes there exists a cardiac crisis, analogous to the "crises gastriques," resembling only remotely true stenocardia. Other clinicians, notably Huchard, connect the stenocardia of tabetics with arterio-sclerosis, which in these patients is of frequent occurrence. The latter is looked upon by others as a dystrophy of the aorta and its valves, similar to "mal perforant" and osteoarthropathy. This latter theory cannot be denied, considering that long-continued forms of neuralgia and neuritis may lead to arterio-sclerosis in the involved area. The neuritic arterio-sclerosis, produced experimentally, by Alexander Fraenkel (Vienna) should also be judged from this point of view. For our purpose those cases are of special importance in which aortic dilatation on an atheromatous basis are present in patients with reflex immobility of the pupils. Sometimes these cases are complicated by lightning-like pains and ataxia in the upper extremities. When one considers that the fibers of conduction of the ciliospinal center leave the spinal cord at the anterior root of the first dorsal nerve and connect with the sympathetic by a communicating branch and, again, that the dorsal nerves send sympathetic branches to the thoracic and abdominal plexuses, the thought cannot be repressed that the communicating rami of the corresponding spinal section contain fibers which are capable of various functions. It is not impossible that a lesion of this tract may lead to angina pectoris or even to arterio-sclerosis. In this connection a case of angina pectoris, communicated by Debove, is of special interest. Debove observed a patient who experienced severe precordial pain together with the girdle sensation, the pain radiating along the left arm down to the little finger. Heart as well as aorta appeared healthy. Two years after this stenocardiac attack there occurred immobility of the pupils, Westphal's phenomenon and lightning pains. This was then a case of angina pectoris appearing as an early symptom of tabes.

Syphilis.—Angina pectoris with syphilis occupies a special position among the forms of stenocardia which are associated with organic lesions. Considering the relations of syphilis to the vessels and to arterio-sclerosis, it is not surprising that in a large number of cases, stenocardia in syphilitics is a form of true coronary angina. In the same way gummous or tuberculo-syphilitic forms of myocarditis will manifest themselves as true coronary angina if they occupy certain positions in the heart, as the area of the coronary arteries. Much more important—because susceptible to antisyphilitic treatment—are those forms of angina pectoris which occur in the early stages of syphilis and lend a peculiar stamp to the stenocardiac picture by their association with other symptoms. Vitono mentions a patient 34 years of age who, four years after acquiring syphilis, presented normal cardiac findings, but typical paroxysms of precordial pain, radiating toward the left arm, and pallor of the skin, following muscular exertions. During some of the attacks, however, there were vasomotor disturbances on the entire left side of the body, sensation of heat and hyperhydrosis.

Treatment.—Sublimate injections. After the sixth injection the paroxysms ceased; recovery followed.

Fournier cured a young man of angina pectoris in the secondary stage of syphilis by pills containing protoiodid hydrargi.

A case of Hallopeau is very instructive. A man 36 years of age, not neuropathic, neither drinker nor smoker, contracted in 1867 a hard chancre followed by papular syphilis. Specific treatment. Eleven years after the primary affection there was suddenly, at night, an attack of angina pectoris (oppression, pains radiating toward the left shoulder and the nucha, in later attacks extending also to the arm and the last three fingers). In the succeeding attacks vasomotor manifestations occupied the foreground: feeling of cold in the left cranial hemisphere, later very cold sensation in the entire left half of the body. While in the further course of the disease both frequency and intensity of the stenocardiac attacks decreased, the patient now manifested paræsthesia and paresis of the right leg, also partial recrudescence of the original trouble which gave rise to the stenocardiac paroxysms and the spread of the same to other organs, such as is frequently observed in syphilitic cases. Upon the administration of 3.0 grams of potassium iodid per day there was complete cessation of the stenocardiac paroxysms, and after further treatment a complete recovery resulted without relapse. Hallopeau attributes the cause of the trouble to an affection of the sympathetic fibers of the cardiac plexus which in the first instance caused the angina pectoris, and, as the lesion spread to other fibers of the plexus, produced new and extensive vasomotor disturbances of the entire left half of the body, with simultaneous recrudescence of the initial lesion.

The occurrence of paræsthesia and paresis of the right leg points to a multiplicity of lesions, such as are frequently observed in syphilis of the central nervous system.

Benenati recently reported quite a series of observations from the clinic at Naples in which changes in the smallest vessels of the cardiac plexus were found, and called attention to the assumed fact that the original seat in many cases of syphilitic angina pectoris is not to be found in the aorta and coronary arteries, but in the aortic or coronary plexus. The rapid effect of antisyphilitic treatment would be in agreement with this assumption.

III

FUNCTIONAL FORMS OF ANGINA PECTORIS

A TRANSITION from the forms of stenocardia dependent upon organic changes in the vessels and the cardiac nerves, to the non-organic, functional forms of angina, includes those forms which occur in arthritic and diabetic patients.

Gout.—Those authors who consider themselves justified in assuming the independent existence of an angina arthritica, point to the fact that this form alternates with other manifestations, such as podagra and cheiragra, also to the occurrence of the attacks after midnight without previous muscular exertion; their cessation in the early morning hours and the disappearance of the attacks upon the appearance of the articular affection.

Diabetes.—Just as incomplete are the investigations of stenocardia in diabetes which occurs also in patients with no demonstrable changes of the vessels, and is said to recede with the improvement of the diabetes. These forms of angina in arthritic and diabetic patients may be of toxic, neuritic or organic origin, especially as these affections constitute a predisposing factor for arterio-sclerosis.

Lead Poisoning.—The same is true of angina in chronic lead poisoning, the relations of which to gout and arterio-sclerosis are well known. Stenocardia in Basedow's disease should be considered from the same point of view, because also in these cases the purely vasomotor element, the neuritic, or the organic, may occupy the foreground of the picture.

Pseudo-anginas.—The second principal group is made up of those forms of angina which are not dependent upon organic lesions of the vessels, but belong to the class of purely functional disturbances. The nervous, hysteric, reflex, partly also the toxic, and especially the tobacco forms of angina belong in this group. The majority of these forms have only in a few features any similarity with true stenocardia. The neuralgic element as well as other nervous symptoms prevail in the pathological picture as opposed to the grave condition of cardiac collapse which characterizes the paroxysms of true stenocardia. These forms of angina are, therefore, also designated false or pseudo-angina. A special group is formed by the so-called reflex-anginas. The reflex may proceed from various organs. Neuralgia of the peripheral nerves, as for instance the intercostal nerves, severe neuralgia in both arms, neuroma, neuritis, neuralgia in stumps after amputation of limbs, especially in those of the left upper extremity, have frequently been mentioned as causative factors in angina pectoris. Frequently angina is occasioned by an affection of the abdominal organs. Dyspepsia, gastric ulcer, perigastritis, gallstones, enterocolitis, especially in the flexura coli lienalis, affections of the female genitals, are some of the numerous points of origin mentioned in the literature as responsible for stenocardia. Anginas occurring at the time of the menopause, forming part of the climacteric heart troubles, also belong to this group.

Epilepsy and Stenocardia.—Many forms of abortive epilepsy, accompanied by pains in the cardiac region which radiate to the left arm, with pallor of the face and extremities, may have a superficial resemblance to stenocardia. On the other hand, epilepsy in patients suffering from cardiac insufficiency may present the character of stenocardia both during the aura and in the post-epileptic state. Stintzing calls attention to cases of mitral stenosis where short stenocardiac paroxysms develop following after an epileptic attack, as consciousness returns. In a similar way grave cardiac affections may impart a peculiar feature to hysteric attacks. Angina pectoris, in hysterical patients suffering from cardiac disease, may cause unsurmountable difficulties in diagnosis. The establishment of the etiology of angina pectoris is in many cases difficult, for the reason that several etiological factors may be jointly active in one and the same individual, as is apparent from the example just cited.

Tobacco Angina.—The type of a toxic angina is the tobacco angina. Abuse of tobacco may also lead to arterio-sclerosis and thus produce coronary angina. In Huchard's clinic an inveterate smoker, who suffered from stenocardia, suddenly expired. At autopsy an atheromatous plaque was discovered at the junction of the coronary arteries.

Infectious Diseases.—Again, in angina occurring after infectious diseases as influenza, there may be several etiological factors. The same is true of angina pectoris following malaria, the attacks of which were treated and cured by quinine. Stenocardia in malaria is associated with endarteritis malarica by a few authors. On the other hand, it is a remarkable fact that Lavaron has never observed angina pectoris in malaria.

IV

DIFFERENTIAL DIAGNOSIS OF ANGINA PECTORIS

The diagnosis of angina pectoris has two objects: In the first place it has to establish that, as a matter of fact, there is a stenocardiac paroxysm, and in the second place it has to determine its cause. The first task, in the majority of cases, is easy, but in rudimentary cases it may occasion considerable difficulty. It should not be forgotten that the very gravest cases often appear indistinct in the first stages. The sudden onset, the intensity, the retrosternal seat of the pain, its radiation, especially to the left arm, the absence of dyspnœa, the prostration of the patient, the short duration of the attacks, the distress and the pallor of the face during the attacks, the relative euphoria during the free intervals—all these points characterize the typical stenocardiac paroxysm.

Asthma.—In bronchial, as well as in cardiac, asthma, dyspnœa with bronchial râles prevails, frequently characterized by the impossibility of deep inspiration, by fixation of the thorax in the position of inspiration, also by cyanosis in cardiac asthma. The patient, even with considerable dyspnœa, will leave the bed to run to the window to get a breath of fresh air, which is very different from patients during stenocardiac attacks, who anxiously avoid the slightest movement. Respiration is sometimes accompanied by distinct râles, sibilant or snoring sounds, which are audible at a distance. As the dyspnœa increases there is an oppression, of the chest in asthma, and it is just this oppression, regarded as anguish, which obscures in many cases the differential diagnosis between asthma and stenocardia.

Pressure Dyspnœa.—The same is true for the paroxysms of acute dyspnœa in compression of the trachea or of a principal bronchus, in occlusion of the same by a foreign body, in embolus of the pulmonary artery and in acute pericarditis. In many cases dyspnœa and cyanosis form the characteristic diagnostic factor in differentiation from angina pectoris. The simultaneous occurrence of cardiac asthma and angina pectoris, however, is no rarity, and in such a case nothing but the sequence and the development of symptoms can save from a mistaken diagnosis. The pallor of the patient during a stenocardiac attack is the rule; in protracted attacks death may supervene with dilatation of the heart and intense cyanosis, but not in an attack of syncope. On the other hand, in thrombosis of the pulmonary artery, such as occurs in carcinoma, tuberculosis, gastric ulcer and sometimes even in chlorosis, it may happen that there is an absence of cyanosis during the entire course of the illness until death: a fact which should not escape attention in the differential diagnosis of complicated cases. The relatively short duration distinguishes, as a rule, the stenocardiac paroxysm from cardiac asthma, but here again there are enough exceptions.

Neuralgias.—Thoraco-brachial neuralgia is characterized by pressure-points, the superficial seat of the pain, paroxysmal exacerbations and increased pain on movement. It should not be forgotten, however, that this kind of brachial neuralgia may precede the true paroxysms of angina pectoris, as happened in a case of Loewenfeld in which the autopsy disclosed atrophy and sclerosis of the nerve trunks of the left brachial plexus. On the other hand, brachialgia may persist after paroxysms of stenocardia. A case is known to me where herpes zoster of the upper extremities developed after coronary angina. The same is true of intercostal neuralgia, both the pressure-points and the skin hyperæsthesia are diagnostically important. The diagnosis may become more difficult in the absence of the pressure-points; when there is intercostal neuralgia as a sequel of aneurysm, even accompanied by herpes zoster; and when coronary angina is accompanied by hyperæsthesia of the skin, as was the case in a patient of Lepine in whom autopsy revealed an occlusion of the anterior coronary artery.

Phrenic neuralgia is observed in many forms of pleuritis (diaphragmatica), pericarditis, aortitis and dissecting aneurysms, and is characterized by dyspnœa, later singultus, sensation of constriction at the insertion of the diaphragm and at the neck, frequently by the radiation of the pain to the left arm. The physical signs in the thoracic organs which arise from a pericarditic or pleuritic exudate, perhaps accompanied by fever, may point out the right way in difficult cases of this kind. On the other hand, it should not be overlooked that the cause of pericarditis in such cases may be found in an affection of the aorta or an aneurysm, and that true stenocardiac paroxysms have also been observed in rare cases in the early stages of pericarditis.

If we have succeeded with some degree of probability, in qualifying a given symptom-complex as angina pectoris, there is now left the task of establishing the etiological diagnosis. The principal problem centers on the question whether or not the case to be diagnosed is one of angina resting upon coronary sclerosis. Age and sex of the patient, the physical examination of the heart, the general impression of the patient, the occurrence of an attack after movements (climbing of stairs), serious affection of the mind, and especially the terrified expression of the patient's face during an attack, and many other manifestations which have already been dwelt upon, will in most cases determine our diagnosis. Here again, however, there are many cases in which an exact diagnosis is impossible, particularly in young persons in whom the examination of the heart reveals no condition whatever deviating from the normal. There is probably no physician who has not, at some time, been taken unaware by the sudden death of a patient during a paroxysm, who may previously have had some complaint, but none of a serious nature.

Hysteria.—The diagnosis of hysterical angina pectoris may sometimes be easy and sometimes exceedingly difficult. The appearance of the patient during the paroxysm may be pale and show suffering. The coldness of the extremities, the sensation of cold, the shivering, and the cold perspiration may suggest coronary angina. In other cases the complaints about cardiac pain predominate, the complexion and expression of the face being unchanged. These cases should unquestionably be designated as functional angina. Aside from the cardinal symptoms of stenocardia we find, as is frequently the case in hysteria, an abundance of secondary manifestations, as spasm of the diaphragm, dyspnœa with considerable acceleration of the respiration, singultus, palpitation of the heart, amblyopia, aphonia, nausea, vomiting, tenesmus, metereorismus, nervous coughing, globus, intercostal pain, skin hyperæsthesia in the precordial region, ovaralgia, hemianæsthesia, at the end of the attack laughing and weeping, eructation, flatus, spastic urination, contractions, and frequently persistent pain at certain points. The retrosternal pain in many cases does not consist in a sensation of narrowness and contraction, but in that of fullness and distension of the thorax. The paroxysms are frequently spontaneous, or follow after excitement. The factors which cause or give rise to them are very variable; frequently the attacks occur at certain hours, or several times in the day, without affecting the general wellbeing; the duration of the attacks varies considerably, usually they last three hours or longer. While typical coronary angina in the majority of cases shows a modest simplicity in its symptom-complex, hysterical angina presents an abundant multiplicity of symptoms, changeability in the radiation of pain, and a misproportion between subjective sensation and objective signs. While patients suffering from typical coronary angina anxiously avoid every word and movement, those with hysterical angina are usually found noisy and lamenting: there is more noise than danger. The diagnosis, however, may become more difficult if hysteria develops in patients suffering from cardiac affections; here the heart presents, as it were, the *locus minoris resistantiæ* for the discharge of hysterical attacks. The most difficult problems may be presented by the diagnosis in hysterical men who suffer from a simultaneous affection of the aorta. Some particular hysterical feature may point the way in these cases to a correct diagnosis, as for instance the complete anæsthesia of the left arm after an attack; the constriction of the field of vision; the occurrence of contractures; the very fact that the attacks do not occur after movements of the body but in a state of rest, and that in spite of their frequency the general well-being is not affected. This explains, then, that in the diagnosis of stenocardia an important guide for the classification of a given case may be furnished by the conditions which precede or follow the attack. Of interest are also those forms of hysterical angina which occur in young patients whose parents have succumbed to a stenocardiac attack, even when they were not present at the death-bed and where the angina, as for instance in the case of young girls, occurs only at the time of puberty.

Neurasthenia.—A special variety of the functional form is the pseudo-angina of neurasthenics, frequently met with in anæmic persons, and rather often, too, in physicians who anxiously auscultate themselves. Here it is less a question of angina pectoris than of angina-phobia with all kinds of abnormal sensations in the cardiac region, along the spinal column, feeling of heaviness on the chest, distension of the heart, dysphagia, difficulty in swallowing, pain in the back of the neck and the shoulders: symptoms in which the psychic element plays the chief rôle and where the mere thought of the disease and its treatment is sufficient to provoke an attack.

Angina Pectoris Vasomotoria.—The consideration of hysterical angina leads on to those forms which have been designated angina pectoris vasomotoria by Eulenburg, Lindois and Nothnagel. They seem to be a part manifestation of a widely diffused vascular spasm. There is an extreme pallor of the face and ears, coldness of the extremities, decrease of sensitiveness, vertigo, fainting attacks, which usually precede the stenocardiac pains and palpitation. The causative element is usually cold, as for instance sleeping in a cold room, washing the hands in cold water. The patients may prevent the palpitation and distress by rubbing their hands and feet, and walking about gives relief, all of which is in opposition to the conditions in true angina pectoris.

Reflex Anginas.—If the stenocardiac attacks do not occur after exertion, nor spontaneously during the night, but at a certain time after a meal, then there is reason for taking the possibility of a so-called reflex angina into consideration. The paroxysms commence with an unpleasant sensation in the epigastrium, oppression, dyspnœa, violent palpation, arrhythmia, inclination to fainting. There are signs of dyspepsia or distension of the stomach and, in the interval, symptoms of a gastric affection, as vomiting, eructation, etc. In old persons who suffer from fatty heart the differential diagnosis between coronary angina and a gastric form, as just described, may occasion difficulty, especially in cases where acute indigestion is followed by grave manifestations on the part of the heart, such as bradycardia or arrhythmia. Calomel may sometimes be of use.

Gastric Ulcer.—In youthful patients gastric ulcer may give rise to considerable difficulty. Last year a female patient, 33 years of age, with healed tuberculosis of the left pulmonary apex and an accentuated second aortic sound, followed by a short dystolic murmur, was received in our clinic. She was somewhat emaciated and pale, the abdomen distended, and below the xyphoid process a painful spot could be palpated. The chief complaint of the patient was about pains which began in the left side of the thorax, progressed retrosternally upward and extended to both arms. The pains occurred particularly when the patient was walking about or did some heavy work. She states there was considerable perspiration during the attacks and also palpitation followed by vomiting, after which relief came. The vomited matter consisted of the ingested food or of a bitter, clear fluid. She had to open her dress during the attack, which after a period of about ten minutes ceased. The retrosternal pains and the radiation into the arms increased in severity and there were continuous attacks for two months, until the patient vomited a large quantity of blood during an attack in which the pain in the gastric region was excessive, after which she fainted away. During her stay in the clinic both pulse frequency and temperature were normal. Our diagnosis, therefore, was: gastric ulcer, insufficiency of the aortic valves, tuberculosis of the left apex of the lungs, reflex angina pectoris.

Traube had already pointed out that gastric ulcers may cause severe attacks of oppression of the thorax without any of the commonly associated pain, so that the oppression forms the chief complaint. This is explained by the proximity of a perigastritis to the solar plexus and the relation of the latter to the dorsal nerves through the splanchnic, and to the cervical nerves through the phrenic. In this way there may occur painful attacks with radiation toward the arms, shoulders, back of the neck and even the maxillary region, which may bear the character of stenocardiac pains.

As a counterpart to the above case the following anamnesis may be cited:

On October 29, 1903, a woman 26 years of age was admitted to our clinic. Up to two months ago she had been tolerably healthy and able to work. It was only recently that in rapid walking she experienced pain in the upper thoracic region which became so severe that she was forced to stand still. The pain did not occur suddenly, but came gradually, then extended backward toward the spine and left arm. When she stretched out the arm, the pain subsided somewhat. Respiration was unimpeded during the attack. The patient felt only that the heart beat was rapid. In a recumbent or sitting posture this did not happen. Whenever she walked on the level for five minutes, or went up a few steps, there was palpitation, then gradually pains were experienced in the chest and back, so that she had to stand still until the pain slowly subsided. In the last six weeks she was no longer able to do her work and the pains also occurred while she was lying down, but were not continuous. During the attacks the patient felt very hot and there was considerable palpitation. She consulted a physician, who, however, was unable to locate any disease, and advised the patient to eat well, as she looked very emaciated. The physician-in-chief of her sick-fund also declared she was healthy and able to work. But the attacks continued to recur more frequently and the patient was able to walk only with considerable effort. Three weeks ago she again went to a physician, who told her she was chlorotic and had irregular heart sounds. He prescribed iron for her. Last Saturday, that is, October 24, 1903, the patient had exceptionally severe pains in the left side of the chest, which became still more intense upon coughing. The appetite was bad, as was the stool; the urine was abundant. In the afternoon of October 29th she complained of cold, became pale and shivered, and commenced to vomit. The patient recovered from this attack, but there followed another during the night. She vomited all ingesta and complained of great thirst.

Present condition—October 30, 1903. Pulse 128, temperature 36°, respirations 24. Light cyanosis of the face, sensorium completely free, extremities cold, very slight œdema of the knuckles. The lower sternum somewhat sensitive to pressure, voice clear. Nothing abnormal was found in the examination of the lungs. Cardiac impulse in the fifth intercostal space in the mammary line, rather strong and regular. Dullness of the heart to the middle of the sternum. The first mitral sound was somewhat dull, the second pulmonic strong and the second aortic sound pure. The heart action is considerably accelerated, 130-140. At the same time the pulse is very small and hardly palpable, but regular; blood pressure 75 mm. Abdomen not distended, musculature very tense. Liver, spleen and kidneys cannot be palpated. Urine shows traces of albumen and the sediment contains a few hyaline casts. Patellar reflex prompt. The vagus trunks not sensitive to pressure. On October 30, 1903, the patient vomited milk previously ingested. The vomited matter reacted weakly; acid contained neither free hydrochloric acid nor lactic acid. Pulse frequency sank to 100 toward the evening. October 31: Patient feels considerably better, has no nausea and asks for food. On November 1, 2, and 3 there was relative wellbeing and the patient ate with good appetite. During the night of November 3 she went twice to the toilet. She became more and more restless and complained to the nurse of tenesmus. Early in the morning she complained of pain in the abdomen and chest, and was given tea with brandy, which she promptly vomited. She became very restless, flexed and lifted her legs, forcefully stretched her arms above her head and loudly complained of her pains. When spoken to, she evidently understood the question, but her only answer was words of complaint. Highly cyanotic, livid. The cervical veins are much swollen. The extremities are cold. At the radial artery no trace of pulse can be detected. The eyes are tightly closed. Auscultation of the heart reveals a dull first sound and a reinforced second sound. Heart action is moderately accelerated, 90-100, but perfectly regular. Patient is given amylnitrite to inspire. Suddenly she becomes absolutely quiet, the respiration stops, there is opisthotonus, the veins protrude still more markedly, there are no more heart sounds to be heard, she draws a few more breaths and expires.

Autopsy showed the following condition:

Myomalacia of the left ventricle of the heart which has thin walls, also dilatation of the ventricle; endarteritis of the aorta, especially above the aortic valve, with occlusion of the beginning of the anterior coronary artery and stenosis of the beginning of the posterior artery. Disseminated fatty degeneration of the right ventricle with slight enlargement; congestion of the liver, spleen, kidneys and intestinal tract. Œdema of the gall-bladder and of the brain.

From this case it is clear that the clinical picture under which sclerosis of the coronary arteries occurs, may also be manifested under the guise of an abdominal complaint. In our patient the final phase of the disease was characterized by pains in the abdomen and vomiting. We know that nausea and vomiting frequently occur in rupture of the heart. The present case proves that this symptom may also occur following myomalacia of the heart or also following coronary angina which precedes the final manifestations by several weeks, as is shown from the anamnesis. Similarly, aneurysm of the aorta may in consequence of irritation of the pneumogastric nerve induce vomiting and stenocardiac attacks; this refers especially to aneurysms of the descending aorta and the abdominal aorta, where paroxysms of pain resembling those in stenocardiac attacks are likewise observed. As a rule, no striking manifestations are observed on the part of the brain. Vertigo, vomiting, also cephalalgia, even slight delirium, certainly do occur now and then during and after an attack of coronary angina; usually, however, the sensorium is free and the attacks occur with full consciousness. In rare cases, however, there is a loss of consciousness and even epileptiform attacks in coronary angina: manifestations which may have mere mechanical causes. Thus, for instance, turgescence of an aneurysmal sac may cause the occlusion of the starting point of an efferent vessel, as for instance of the atheromatously stenosed carotid, and in the further course of the disease, cerebral anæmia may lead to loss of consciousness and convulsions, particularly if the cerebral arteries are sclerosed and the compensating circulation of the circle of Willis is interfered with, so as to produce an acute ischæmia of the brain.

Tobacco Angina.—The diagnosis of tobacco angina rests upon the behavior of the patient during the attack and during the intervals. The intensity of the vertigo, the intermittence of the pulse, the extreme pallor, the coldness of the extremities, the muscular tremor, the scotoma, the amblyopia, the cephalalgia and the long duration of the attacks may aid the diagnosis. Difficulties, however, present themselves in those cases where two or more injurious factors are at work, thus, for instance, if an excessive smoker shows manifestations of an atheroma of the aorta with accentuation of the second sound and increased vascular tension. In cases of this kind there is nothing to do but to observe the patient's behavior when he discontinues smoking. The duration of the attacks and their frequency may play a rôle in the differential diagnosis.

Status Anginosus.—We know that the typical attack of coronary angina is usually short, but if there is a frequent repetition of the cause, an almost permanent status anginosus may develop. As time goes on, slight provocations may suffice to cause an attack. Possibly the nerves become more sensitive and more easily irritated so that the attacks occur apparently spontaneously, perhaps in the night during sleep, without any preceding excitation or exertion. These nightly attacks are then of long duration and are not infrequently combined with cardiac asthma. In other cases the attacks are provoked less by muscular exertion than by the influence of cold. Thus I have known a case of coronary angina where the mere contact of the fingers with a cold object was sufficient to occasion an attack. The touch of cold bed linen, the low temperature of a room and a cold wind may have the same effect. On the other hand, there are also instances where patients who had previously experienced typical attacks with frequent paroxysms of pain, are only subjected to abortive attacks in the further course of the disease, characterized by short periods of weakness, and yet, even in these abortive attacks, patients may finally succumb. Finally, there is a possibility that attacks of angina pectoris may gradually decrease in frequency and intensity and then disappear altogether. In these cases the patients may still suffer from cardiac insufficiency or succumb to other affections, such as gastric ulcer, without being troubled any longer by stenocardia.

In a case of this kind Curschmann found a complete obliteration of a small branch of the left coronary artery. In other cases it is possible that a collateral circulation through anastomosis has been established, as was demonstrated by Langer and Toldt. When there are adhesions between the heart and pericardium, branches of the arteria pericardico-phrenica, or pericardica posterior, may, according to Hyrtl, penetrate the muscular structure and anastomose with the branches of the coronary arteries. These latter, as Hyrtl especially emphasizes, may again combine with the bronchial arteries.

Prognosis.—We have already called attention to the fact that angina pectoris may, after a short course, lead to sudden death in patients with cardiac affections. The frequent occurrence of grave attacks in rapid succession especially suggests the highly probable prognosis of a rapidly fatal end. In other cases the attacks repeat themselves at very long intervals, and finally the patient suddenly dies during one of the attacks. Sometimes the angina disappears, although the cardiac trouble continues, so that the patient may finally succumb to inefficiency of the cardiac function. This constitutes the so-called self-cure to which reference has already been made. Finally, angina pectoris may be entirely cured, as Curschmann's cases, as above cited, distinctly prove.

V.

THEORIES OF ANGINA PECTORIS

Arterial Theory.—The uncertainty of prognosis in angina pectoris lies in part in the fact that we are not yet sufficiently informed about the mechanism of the attacks and the cause of death. The majority of clinicians hold to the arterial theory and look to ischæmia of the mycocardium for the key to explain all the manifestations of a stenocardiac attack. Plausible though this theory is in the attempt to explain an organic coronary angina, it does not provide a satisfactory explanation of cases where patients die during the attack and the autopsy reveals no anatomical lesion of the coronary vessels whatever.

Nerve Theory.—This objection led to the formation of the nerve theory, which assumes that inflammatory processes of the cardiac plexus are the cause of the affection, and this was in many cases supported by the fact that lesions were found in the nerves of the cardiac plexus. If we consider that both vasoconstricting and vasodilating fibers of the coronary area terminate in the cardiac plexus and that these fibers connect with the spinal cord through the intermediary of the sympathetic ganglia and the rami communicantes; if we keep in mind that the excitations, which start from the centers for the vascular musculature, proceed from the gray substance to the anterior roots and from there through the rami communicantes to the sympathetic, the possibility suggests itself that the sensory fibers carry the sensation of pain in a centripetal direction, whereas the excitation of the centrifugal fibers is manifested as a vascular spasm in the cardiac vessels. This would complete a circle, in which not only primary excitation, but also reflex processes may be manifested in the course of the rami communicantes through the connection of the spinal cord with the sympathetic. This system also includes the pneumogastric nerve. The investigations of Maass have shown that vasoconstrictor fibers for the coronary artery terminate in the pneumogastric nerve, and since it is known that the rami communicantes effect the connection of the sympathicus with the cervicobrachial plexus, a typical stenocardiac attack could be explained by any irritation of that system. In order to bring about a stenocardiac attack it would therefore be a matter of indifference whether the causative irritation starts in the end apparatus—that is the sensitive organs of the coronary arteries—in the transit station—that is the cardiac nerves—in the rami communicantes, or finally in the central apparatus—that is the spinal cord. The irritation of the sensitive connections would therefore produce pain, and that of the centrifugal connections (vasomotor) ischæmia of the myocardium. According to which of these components prevails, the polymorphic character of the stenocardiac paroxysms and the phenomena of radiation would be susceptible of interpretation. The anguish could be explained as being due to the centripetal connections and to the vagus, owing to an irritation of the oblongata. Let us keep the complete circle before our eyes: the spinal center of innervation, the dorsal nerves, the rami communicantes, the cardiac nerves, the aortic plexus and coronary plexus with the sensitive terminal organs in their vascular walls. We shall be able to trace to one common basis all the previously discussed types of stenocardia, much though they may differ in their anatomical foundation. Any and every irritation within that circle may cause alarm in the heart. In this way spinal affections (tabes), injuries to the dorsal nerves and the rami communicantes (case of Wallenberg), affections of the nervus cardiacus magnus, neuritic affections of the aortic and coronary plexus, and finally embolism and thrombosis of the coronary arteries, may produce the symptom-complex of angina pectoris. The result will be the same in all cases; in the center pain and anguish, in the periphery vascular spasm with ischæmia of the myocardium. A healthy heart will overcome such an attack, even though it be serious, but if the heart muscle is weak and reduced in efficiency, whether from deficient nutrition or fatty degeneration, myocarditis or adhesions to the pericardium, then even a rudimentary vascular spasm in the heart muscle may lead to a catastrophe, just as a fatty degenerated heart may suddenly cease to work without the presence of an acute ischæmia.

VI

THERAPY OF ANGINA PECTORIS

The therapy of angina pectoris should above all be an etiologic one: syphilis, gout, lead poisoning, malaria and diabetes should not be overlooked. In these forms treatment may be of some avail. Unfortunately, the most frequent cause of angina pectoris is arterio-sclerosis, and against this affection we are as yet powerless. The relation of the affections to the adrenals is known. I may refer to two cases which I have mentioned already in my work on the affections of the adrenals. They refer to young persons, one 25 years of age, with wiry, tense pulse and multiple hemorrhages of the brain, so that during life the diagnosis of Bright's disease was made. Autopsy showed a carcinoma of the suprarenal capsule. What is more feasible than the assumption of a pathologically increased secretion of the adrenal products which will raise the blood pressure? Endarteritic vascular affections have repeatedly been observed in young persons with adrenal affections for which no other cause was assignable, so that it is quite probable that there is an intimate connection between the chromaffin system and arterio-sclerosis.

A hypersecretion of the chromaffin cells might cause not only the increased blood pressure, but also, through vascular spasm of the smallest vasa vasorum, secondary anatomical changes, just as is observed in other poisons, for instance in ergotin. Perhaps this opens up an avenue for the prophylaxis and therapy of hypertension, since it is not impossible *a priori* to produce efficacious antibodies by transplanting the adrenal substances into suitable parts of the body. We are now, however, limited to a symptomatic therapy in the treatment of angina pectoris, without being able to place great hopes upon it, for even the favorite iodin preparations nearly always fail us, except when syphilis is a factor.

The therapy of the attacks is antispasmodic, to increase the flow of blood through the myocardium by dilatation of the coronary vessels, thus reinforcing the cardiac action against the increased pressure of the aorta and improving the heart function by an increased blood supply. In the first place the nitrites, such as natrium nitrosum, amylnitrite, nitroglycerin and erythroltetranitrate. The theobromine preparations not only improve the heart action, but probably also have a vasodilating effect in the area of the cardiac arteries. Quinine also, administered in small doses for some length of time, renders frequently good service, and this personal experience of mine agrees with Hedbom's experiments upon animals, according to which there was an increased circulation after the administration of quinine. Generally speaking, the success of the present therapy should not be overestimated, knowing, as we do, that the attacks of angina pectoris may cease spontaneously for long-continued periods. Only in the employment of digitalis—the sovereign remedy in the treatment of heart affection—the utmost caution should be employed, as it contains substances which have a vasoconstrictor effect. The same applies to morphin, as we have already pointed out.

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